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=> fil reg
COST IN U.S. DOLLARS                                SINCE FILE      TOTAL
                                                       ENTRY    SESSION
FULL ESTIMATED COST                                    62.32      84.84

DISCOUNT AMOUNTS (FOR QUALIFYING ACCOUNTS)          SINCE FILE      TOTAL
                                                       ENTRY    SESSION
CA SUBSCRIBER PRICE                                    -2.35      -2.35

FILE 'REGISTRY' ENTERED AT 22:51:04 ON 25 NOV 2001
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PLEASE SEE "HELP USAGETERMS" FOR DETAILS.
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STRUCTURE FILE UPDATES:   23 NOV 2001  HIGHEST RN 371752-43-1
DICTIONARY FILE UPDATES:  23 NOV 2001  HIGHEST RN 371752-43-1

TSCA INFORMATION NOW CURRENT THROUGH July 7, 2001

  Please note that search-term pricing does apply when
  conducting SmartSELECT searches.

Crossover limits have been increased.  See HELP CROSSOVER for details.

Calculated physical property data is now available.  See HELP PROPERTIES
for more information.  See STNote 27, Searching Properties in the CAS
Registry File, for complete details:
http://www.cas.org/ONLINE/STN/STNOTES/stnotes27.pdf

=> s 566-19-8
L7            1 566-19-8
                (566-19-8/RN)

=> d

L7   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2001 ACS
RN   566-19-8  REGISTRY
CN   Androst-5-ene-7,17-dione, 3-hydroxy-, (3β)- (9CI)  (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN   Androst-5-ene-7,17-dione, 3β-hydroxy- (8CI)
OTHER NAMES:
CN   3β-Hydroxy-5-androstene-7,17-dione
CN   5-Androsten-3β-ol-7,17-dione
CN   7-Ketodehydroepiandrosterone
CN   7-Oxodehydroepiandrosterone
FS   STEREOSEARCH
MF   C19 H26 O3
LC   STN Files:   BEILSTEIN*, BIOSIS, CA, CAOLD, CAPLUS, CASREACT, IFICDB,
       IFIPAT, IFIUDB, MEDLINE, TOXCENTER, TOXLIT, USPATFULL
         (*File contains numerically searchable property data)

Absolute stereochemistry.
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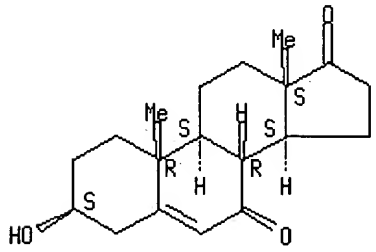


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**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

             69 REFERENCES IN FILE CA (1967 TO DATE)
              6 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
             69 REFERENCES IN FILE CAPLUS (1967 TO DATE)
              2 REFERENCES IN FILE CAOLD (PRIOR TO 1967)
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=> s 1449-61-2
L8            1 1449-61-2
                (1449-61-2/RN)

=> d

L8   ANSWER 1 OF 1  REGISTRY  COPYRIGHT 2001 ACS
RN   1449-61-2  REGISTRY
CN   Androst-5-ene-7,17-dione, 3-(acetyloxy)-, (3β)- (9CI)  (CA INDEX
     NAME)
OTHER CA INDEX NAMES:
CN   Androst-5-ene-7,17-dione, 3β-hydroxy-, acetate (6CI, 7CI, 8CI)
OTHER NAMES:
CN   3β-Acetoxy-5-androsten-7,17-dione
CN   3β-Acetoxyandrost-5-ene-7,17-dione
CN   5-Androsten-3β-ol-7,17-dione acetate
FS   STEREOSEARCH
MF   C21 H28 O4
LC   STN Files:   BEILSTEIN*, CA, CAOLD, CAPLUS, CASREACT, CHEMCATS, IFICDB,
       IFIPAT, IFIUDB, TOXCENTER, TOXLIT, USPATFULL
         (*File contains numerically searchable property data)

Absolute stereochemistry.
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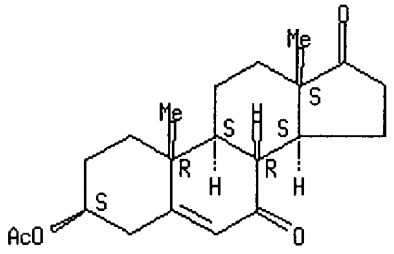


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**PROPERTY DATA AVAILABLE IN THE 'PROP' FORMAT**

              52 REFERENCES IN FILE CA (1967 TO DATE)
              52 REFERENCES IN FILE CAPLUS (1967 TO DATE)
              14 REFERENCES IN FILE CAOLD (PRIOR TO 1967)


=> sel name l7 1
E1 THROUGH E4 ASSIGNED

=> sel name l8 1
E5 THROUGH E7 ASSIGNED

=> fil medlin capl biosis uspatfull

=> s l7-8 or e1-7
   3 FILES SEARCHED...
L9          157 (L7 OR L8) OR ("3.BETA.-HYDROXY-5-ANDROSTENE-7,17-DIONE"/BI
                OR "5-ANDROSTEN-3.BETA.-OL-7,17-DIONE"/BI OR 7-KETODEHYDROEPIAND
                ROSTERONE/BI OR 7-OXODEHYDROEPIANDROSTERONE/BI OR "3.BETA.-ACETO
                XY-5-ANDROSTEN-7,17-DIONE"/BI OR "3.BETA.-ACETOXYANDROST-5-ENE-7
                ,17-DIONE"/BI OR "5-ANDROSTEN-3.BETA.-OL-7,17-DIONE ACETATE"/BI)


=> s ?arthr? or fibrom?
L10      584492 ?ARTHR? OR FIBROM?

=> s l9 (s) l10
L11           1 L9 (S) L10

=> d

L11  ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2001 ACS
Full-text
AN   1999:344832  CAPLUS
DN   131:1145
```

```
TI   Use of Δ5-androstene-3β-ol-7,17-dione in the treatment of
     arthritis
IN   Weeks, Charles E.
PA   Humanetics Corporation, USA
SO   PCT Int. Appl., 17 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     ---------------   ----  --------       ---------------  --------
PI   WO 9925192         A1   19990527       WO 1998-US24458  19981117
         W:  AU, CA, US
         RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
             PT, SE
     AU 9914142         A1   19990607       AU 1999-14142    19981117
     EP 1032266         A1   20000906       EP 1998-958020   19981117
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, FI
PRAI US 1997-66197      P    19971119
     WO 1998-US24458    W    19981117
RE.CNT  2
RE
(1) Lardy, H; US 5585371 A 1996 CAPLUS
(2) Peat, R; US 4528052 A 1986 CAPLUS


=> s l9 and l10
L12          1 L9 AND L10

=> d

L12  ANSWER 1 OF 1  CAPLUS  COPYRIGHT 2001 ACS
Full-text
AN   1999:344832  CAPLUS
DN   131:1145
TI   Use of Δ5-androstene-3β-ol-7,17-dione in the treatment of
     arthritis
IN   Weeks, Charles E.
PA   Humanetics Corporation, USA
SO   PCT Int. Appl., 17 pp.
     CODEN: PIXXD2
DT   Patent
LA   English
FAN.CNT 1
     PATENT NO.        KIND  DATE           APPLICATION NO.  DATE
     ---------------   ----  --------       ---------------  --------
PI   WO 9925192         A1   19990527       WO 1998-US24458  19981117
         W:  AU, CA, US
         RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL,
             PT, SE
     AU 9914142         A1   19990607       AU 1999-14142    19981117
     EP 1032266         A1   20000906       EP 1998-958020   19981117
         R:  AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT,
             IE, FI
PRAI US 1997-66197      P    19971119
     WO 1998-US24458    W    19981117
RE.CNT  2
RE
(1) Lardy, H; US 5585371 A 1996 CAPLUS
(2) Peat, R; US 4528052 A 1986 CAPLUS


=> s ?inflamm?
L13     643305 ?INFLAMM?

=> s l13 and l9
L14          3 L13 AND L9

=> dup rem l14
PROCESSING COMPLETED FOR L14
L15           3 DUP REM L14 (0 DUPLICATES REMOVED)

=> s l15 not l12
L16          3 L15 NOT L12
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=> d tot

L16 ANSWER 1 OF 3 CAPLUS COPYRIGHT 2001 ACS
Full-text
AN 1999:350591 CAPLUS
DN 131:1146
TI Use of Δ5-androstene-3β-ol-7,17-dione in the treatment of lupus erythematosus
IN Lardy, Henry A.; Weeks, Charles E.
PA Humanetics Corporation, USA
SO PCT Int. Appl., 18 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9925333	A1	19990527	WO 1998-US23386	19981103
	W: AU, CA, JP, US				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	AU 9913017	A1	19990607	AU 1999-13017	19981103
	AU 738136	B2	20010906		
	EP 1032380	A1	20000906	EP 1998-956509	19981103
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, SI, LT, LV, FI, RO				
PRAI	US 1997-66198	P	19971119		
	WO 1998-US23386	W	19981103		

L16 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2001 ACS
Full-text
AN 1967:11129 CAPLUS
DN 66:11129
TI 7,7-gem-Difluoro steroids of the androstane and pregnane series
IN Boswell, George A., Jr.
PA du Pont de Nemours, E. I., and Co.
SO U.S., 9 pp.
CODEN: USXXAM
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	US 3282969		19661101	US	19640702

L16 ANSWER 3 OF 3 USPATFULL
Full-text
AN 96:91831 USPATFULL
TI Vaccine compositions and method for enhancing an immune response
IN Daynes, Raymond A., Park City, UT, United States
Araneo, Barbara A., Salt Lake City, UT, United States
PA University of Utah Research Foundation, Salt Lake City, UT, United States (U.S. corporation)
PI US 5562910 19961008
AI US 1993-123843 19930909 (8)
RLI Continuation-in-part of Ser. No. US 1993-13972, filed on 4 Feb 1993, now abandoned And a continuation-in-part of Ser. No. US 1991-779499, filed on 18 Oct 1991, now abandoned which is a continuation-in-part of Ser. No. US 1989-412270, filed on 25 Sep 1989, now abandoned
DT Utility
FS Granted
LN.CNT 1591
INCL INCLM: 424/278.100
INCLS: 424/209.100; 424/211.100; 424/217.100; 424/219.100; 424/224.100; 424/225.100; 424/230.100; 424/231.100; 424/244.100; 424/245.100; 424/247.100; 424/254.100; 424/256.100; 514/169.000; 514/725.000; 514/885.000; 514/171.000; 514/178.000; 514/167.000
NCL NCLM: 424/278.100
NCLS: 424/209.100; 424/211.100; 424/217.100; 424/219.100; 424/224.100; 424/225.100; 424/230.100; 424/231.100; 424/244.100; 424/245.100; 424/247.100; 424/254.100; 424/256.100; 514/167.000; 514/169.000; 514/171.000; 514/178.000; 514/725.000; 514/885.000
IC [6]
ICM: A61K039-00

ICS: A61K031-56; A61K031-59
EXF 424/88; 424/85.1; 424/278.1; 424/225.1; 424/209.1; 424/245.1; 424/247.1; 424/254.1; 424/212.1; 424/219.1; 424/217.1; 424/244.1; 424/231.1; 424/211.1; 424/256.1; 424/230.1; 424/224.1; 424/184.1; 424/256.1; 424/230.1; 424/224.1; 424/184.1; 514/885; 514/167; 514/169; 514/178; 514/725; 514/885; 514/171
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

=> d ibib abs kwic 2-3

L16 ANSWER 2 OF 3 CAPLUS COPYRIGHT 2001 ACS
Full-text
ACCESSION NUMBER: 1967:11129 CAPLUS
DOCUMENT NUMBER: 66:11129
TITLE: 7,7-gem-Difluoro steroids of the androstane and pregnane series
INVENTOR(S): Boswell, George A., Jr.
PATENT ASSIGNEE(S): du Pont de Nemours, E. I., and Co.
SOURCE: U.S., 9 pp.
CODEN: USXXAM
DOCUMENT TYPE: Patent
LANGUAGE: English
FAMILY ACC. NUM. COUNT: 1
PATENT INFORMATION:

PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
US 3282969		19661101	US	19640702

AB The title compds. exhibit a high degree of **antiinflammatory** activity. Solid anhyd. Na chromate was added portionwise to 40 g. 3β,17β-diacetoxyandrost-5-ene in 135 ml. HOAc and 74 ml. Ac2O at 30-40° and the mixt. stirred 46 hrs. at this temp. to give 24.9 g. 3β,17β-diacetoxy-5-androsten-7-one (I), m. 225° (MeOH). I (12.3 g.) in 50 ml. glacial HOAc and 50 ml. EtOH was shaken 48 hrs. under 50 psi. H with 1 g. PtO2, the catalyst filtered off, the filtrate evapd. to dryness in vacuo, the residue dissolved in Me2CO, stirred 20 min. at room temp. with 10.3 g. CrO3 in 30 ml. H2O and 3.7 ml. concd. H2SO4, a small amt. MeOH added, the soln. filtered, and the filtrate dild. with H2O to ppt. 9.49 g. 3β,17β-diacetoxyandrostan-7-one (II), m. 187-90°. II (9.5 g.), 100 ml. CH2Cl2, and 1.5 ml. H2O in a corrosion resistant bomb was cooled in a dry ice-Me2CO bath, 180 g. SF4 added, the bomb agitated 16 hrs. at 20° and vented, the volatiles removed in vacuo, the residue dissolved in CH2Cl2, and the soln. evapd. to dryness to give 8.7 g. 3β,17β-diacetoxy-7,7-difluoroandrostane (III), m. 165°, [α] 23 D -6° (c 2.55, CHCl3). Prepd. similarly was 3β,17β-diacetoxy-7,7-difluoroandrostan-11β-ol. III, 15 ml. concd. HCl, and 150 ml. MeOH was refluxed 1 hr., H2O added to turbidity, and the soln. cooled to give 6.05 g. 7,7-difluoroandrostane-3β,17β-diol (IV), m. 185-7° (Me2CO-hexane), [α] 23 D 6° (c 2.85, dioxane). IV (6 g.) was oxidized as above to give 3.68 g. 7,7-difluoroandrostane-3,17-dione (V), m. 192° [α] 22 D 77° (c 2.61, CHCl3). Also prepd. was 7,7-difluoro-1,4-androstadiene-3,17-dione. V (5.5 g.), 5.0 ml. pyrrolidine, and 30 ml. C6H6 was refluxed 3 hrs. with H2O removal and the solvent removed in vacuo to give 7,7-difluoro-3-pyrrolidinyl-2-androsten-17-one (VI). VI (5.5 g.) was dissolved in 200 ml. dry tetrahydrofuran, 13 g. solid LiAl(OBu-tert)3H added portionwise, the soln. stirred at room temp. 48 hrs., poured into 200 ml. cold MeOH contg. 20 ml. glacial HOAc and 10 g. NaOAc, and the mixt. heated 1 hr. on a steam bath and dild. with H2O to ppt. 2.74 g. 7,7-difluoroandrostan-17β-ol-3-one. To the filtrate was added excess NaCl and the mixt. extd. thoroughly with CH2Cl2, and evapd. to give an addnl. 3 g. which did not cryst. The combined solid was dissolved in a little C6H6 and the soln. chromatographed over 100 g. neutral alumina and eluted with 1:1 petr. ether-C6H6 to give 1.52 g. solid. Continued elution with C6H6 gave 1.007 g. solid, and stripping of the column with 3:1 and 1:1 C6H6-Et2O gave 0.77 g. amorphous material. The petr. ether-C6H6 and C6H6 eluates were combined to give 1.542 g. 7,7-difluoroandrostan-17β-ol-3-one, m. 204-5° (Me2CO-hexane), [α] 23 D 11° (c 2.89, acetone). The C6H6-Et2O eluates were combined and recrystd. from Me2CO-hexane to give 0.65 g. 7,7-difluoro-3β-pyrrolidinylandrostan-17β-ol. Prepd. similarly were: 3β-acetoxy-7,7,17,17-tetrafluoroandrostane, m. 155-6.5°,

[α] 23 D -14° (c 2.59, CHCl3); 7,7-difluoro-17α-vinylandrostane-3β,17β-diol; 7,7,17,17-tetrafluoroandrostan-3β-ol, m. 144-52°, [α] 23 D 0° (c 2.00, CHCl3); 7,7,17,17-tetrafluoroandrostan-3-one, m. 246-8°, [α] 23 D -2° (c 2.48, CHCl3); 7,7,17,17-tetrafluoro-1-androsten-3-one, m. 197-9°, [α] 23 D 16° (c 2.52, CHCl3); 3β,17α-diacetoxy-7,7-difluoropregnan-20-one, m. 222.5-4.0°, [α] 23 D (c 2.45, CHCl3); 17α-acetoxy-7,7-difluoropregnan-3β-ol-20-one, m 225-7°, [α] 23 D -28° (c 2.50, CHCl3); 17α-acetoxy-7,7-difluoro-1-pregnene-3,20-dione, m. 220-5°, [α] 23 D 20° (c 2.00, CHCl3); 3β-acetoxy-7,7,20,20-tetrafluoropregnane, m. 182-3°, [α] 23 D -3° (c 2.77, CHCl3); 3β-acetoxy-7,7-difluoropregnan-20-one, m. 168-73°, [α] 23 D 42° (c 2.38, CHCl3); 7,7,20,20-tetrafluoropregnan-3β-ol, m. 165-6°, [α] 23 D 7° (c 2.57, acetone); 7,7,20,20-tetrafluoropregnan-3-one, m. 157.5-8.5°, [α] 23 D 13° (c 2.31, CHCl3); 17β-acetoxy-7,7-difluoroandrostan-3-one, m. 169-71°, [α] 23 D 6° (c 2.34, CHCl3); 17β-acetoxy-7,7-difluoro-1-androsten-3-one, m. 145-8°, [α] 23 D 33° (c 1.88, CHCl3); 17α-acetoxy-7,7-difluoro-1,4-pregnadiene-3,20-dione, m. 243-5°; and 7,7,17,17-tetrafluoro-1,4-androstadien-3-one, m. 243-5°.

ST ANDROSTANES FLUORO; FLUORO PREGNANES; FLUORO ANDROSTANES; **ANTIINFLAMMATORY** GEM DIFLUORO STEROIDS; GEM DIFLUORO STEROIDS **ANTIINFLAMMATORY**; DIFLUORO STEROIDS GEM **ANTIINFLAMMATORY**; STEROIDS **ANTIINFLAMMATORY** GEM DIFLUORO; PREGNANES FLUORO; FLUORO PREGNANES; PREGNANES FLUORO; **ANTIINFLAMMATORY** GEM DIFLUORO STEROIDS; GEM DIFLUORO STEROIDS **ANTIINFLAMMATORY**; DIFLUORO STEROIDS GEM **ANTIINFLAMMATORY**; STEROIDS **ANTIINFLAMMATORY** GEM DIFLUORO; FLUORO ANDROSTANES; ANDROSTANES FLUORO

IT **1449-61-2P** 6748-09-0P 13209-42-2P 13209-43-3P 13209-60-4P
13209-61-5P 13209-62-6P 13209-63-7P 13209-64-8P 13209-65-9P
13209-66-0P 13209-67-1P 13209-68-2P 13209-69-3P 13209-71-7P
13209-72-8P 13209-73-9P 13209-74-0P 13258-27-0P 13258-28-1P
13258-29-2P 13258-30-5P 13258-31-6P 13258-32-7P 13258-34-9P
13258-35-0P 13258-36-1P 13258-37-2P 13258-38-3P 13258-39-4P
13258-40-7P 13258-41-8P 13385-42-7P
RL: SPN (Synthetic preparation); PREP (Preparation)
(prepn. of)

L16 ANSWER 3 OF 3 USPATFULL
Full-text

ACCESSION NUMBER: 96:91831 USPATFULL
TITLE: Vaccine compositions and method for enhancing an immune response
INVENTOR(S): Daynes, Raymond A., Park City, UT, United States
Araneo, Barbara A., Salt Lake City, UT, United States
PATENT ASSIGNEE(S): University of Utah Research Foundation, Salt Lake City, UT, United States (U.S. corporation)

	NUMBER	KIND	DATE
PATENT INFORMATION:	US 5562910		19961008
APPLICATION INFO.:	US 1993-123843		19930909 (8)

DISCLAIMER DATE: 20130909
RELATED APPLN. INFO.: Continuation-in-part of Ser. No. US 1993-13972, filed on 4 Feb 1993, now abandoned And a continuation-in-part of Ser. No. US 1991-779499, filed on 18 Oct 1991, now abandoned which is a continuation-in-part of Ser. No. US 1989-412270, filed on 25 Sep 1989, now abandoned
DOCUMENT TYPE: Utility
FILE SEGMENT: Granted
PRIMARY EXAMINER: Housel, James C.
ASSISTANT EXAMINER: Krsek-Staples, Julie
LEGAL REPRESENTATIVE: Venable, Baetjer, Howard & Civiletti, LLP
NUMBER OF CLAIMS: 36
EXEMPLARY CLAIM: 1
NUMBER OF DRAWINGS: 43 Drawing Figure(s); 18 Drawing Page(s)
LINE COUNT: 1591
CAS INDEXING IS AVAILABLE FOR THIS PATENT.

AB The invention relates to a vaccine which comprises an antigen and an

immune response augmenting agent. The immune response augmenting agent is capable of enhancing T cell lymphokine production. Suitable immune response augmenting agents include, but are not limited to, dehydroepiandrosterone (DHEA) and DHEA-derivatives. Examples of DHEA derivatives include DHEA-sulfate (DHEA-S), 16α-bromo-DHEA, 7-oxo-DHEA, 16α-bromo-DHEA-S and 7-oxo-DHEA-S.

The invention also relates to a method for enhancing a vaccine-induced humoral immune response which comprises administering a vaccine which comprises an antigen and an immunomodulator. The immunomodulator may be an immune response augmenting agent, a lymphoid organ modifying agent or a mixture of the immune response augmenting agent and lymphoid organ modifying agent. Suitable lymphoid organ modifying agents include, but are not limited to, 1,25-dihydroxy Vitamin D, biologically active Vitamin D derivatives which are capable of activating the intracellular Vitamin D receptor, all trans-retinoic acid, retinoic acid derivatives, retinol, retinol derivatives and glucocorticoid. Alternatively, the method for enhancing a vaccine-induced humoral immune response comprises separately administering the immunomodulator and a vaccine containing an antigen.

CAS INDEXING IS AVAILABLE FOR THIS PATENT.
DETD . . . also be capable of providing regulatory information to cells of the immune system. This information, mediated through the activities of **inflammation**-induced tissue cytokines, prostaglandins and other types of biological response modifiers, becomes integrated into the complex equation to control the mechanisms. . .
IT 53-43-0D, DHEA, derivs. 68-26-8D, Retinol, derivs. 302-79-4D, Retinoic acid, derivs. **566-19-8** 651-48-9, DHEA sulfate 4121-96-4 32222-06-3D, 1,25-Dihydroxy vitamin D3, derivs. 75767-22-5 151434-12-7 151434-13-8 177857-48-6
(vaccine comprises antigen and immunomodulator or lymphoid organ modifying agent)

=> s fibromyalg?
L17 4995 FIBROMYALG?

=> s l17 and l9
L18 1 L17 AND L9

=> d

L18 ANSWER 1 OF 1 CAPLUS COPYRIGHT 2001 ACS
Full-text
AN 1999:344832 CAPLUS
DN 131:1145
TI Use of Δ5-androstene-3β-ol-7,17-dione in the treatment of arthritis
IN Weeks, Charles E.
PA Humanetics Corporation, USA
SO PCT Int. Appl., 17 pp.
CODEN: PIXXD2
DT Patent
LA English
FAN.CNT 1

	PATENT NO.	KIND	DATE	APPLICATION NO.	DATE
PI	WO 9925192	A1	19990527	WO 1998-US24458	19981117
	W: AU, CA, US				
	RW: AT, BE, CH, CY, DE, DK, ES, FI, FR, GB, GR, IE, IT, LU, MC, NL, PT, SE				
	AU 9914142	A1	19990607.	AU 1999-14142	19981117
	EP 1032266	A1	20000906	EP 1998-958020	19981117
	R: AT, BE, CH, DE, DK, ES, FR, GB, GR, IT, LI, LU, NL, SE, MC, PT, IE, FI				
PRAI	US 1997-66197	P	19971119		
	WO 1998-US24458	W	19981117		

RE.CNT 2
RE
(1) Lardy, H; US 5585371 A 1996 CAPLUS
(2) Peat, R; US 4528052 A 1986 CAPLUS

=> d his

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     (FILE 'HOME' ENTERED AT 22:31:55 ON 25 NOV 2001)

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L1             4 S GENISTEIN/CN OR DAIDZEIN/CN OR FORMONONETIN/CN OR BIOCHANIN A

     FILE 'MEDLINE, CAPLUS, BIOSIS, USPATFULL' ENTERED AT 22:34:18 ON 25 NOV
     2001
L2         16130 S L1 OR GENISTEIN OR DAIDZEIN OR FORMONONETIN OR BIOCHANIN A
L3        353448 S OBES? OR WEIGH? GAIN OR WEIGH? LOSS? OR OVERWEIGH? OR APPET?
L4            42 S L2 (S) L3
L5            25 DUP REM L4 (17 DUPLICATES REMOVED)
L6            25 FOCUS L5 1-

     FILE 'REGISTRY' ENTERED AT 22:51:04 ON 25 NOV 2001
L7             1 S 566-19-8
L8             1 S 1449-61-2
                 SEL NAME L7 1
                 SEL NAME L8 1

     FILE 'MEDLINE, CAPLUS, BIOSIS, USPATFULL' ENTERED AT 22:53:28 ON 25 NOV
     2001
L9           157 S L7-8 OR E1-7
L10       584492 S ?ARTHR? OR FIBROM?
L11            1 S L9 (S) L10
L12            1 S L9 AND L10
L13       643305 S ?INFLAMM?
L14            3 S L13 AND L9
L15            3 DUP REM L14 (0 DUPLICATES REMOVED)
L16            3 S L15 NOT L12
L17         4995 S FIBROMYALG?
L18            1 S L17 AND L9

=> s l13 and l17
L19          487 L13 AND L17

=> s l13 (s) l17
L20          246 L13 (S) L17

=> s l13 (6a) l17
L21           55 L13 (6A) L17

=> dup rem l21
PROCESSING COMPLETED FOR L21
L22           40 DUP REM L21 (15 DUPLICATES REMOVED)

=> focus
PROCESSING COMPLETED FOR L22
L23           40 FOCUS L22 1-

=> d ibib abs kwic 1-5

L23  ANSWER 1 OF 40  CAPLUS  COPYRIGHT 2001 ACS
Full-text
ACCESSION NUMBER:         2000:162974  CAPLUS
DOCUMENT NUMBER:          132:303169
TITLE:                    Preference for nonsteroidal antiinflammatory drugs
                          over acetaminophen by rheumatic disease patients: a
                          survey of 1,799 patients with osteoarthritis,
                          rheumatoid arthritis, and fibromyalgia
AUTHOR(S):                Wolfe, Frederick; Zhao, Sean; Lane, Nancy
CORPORATE SOURCE:         Arthritis Research Center and University of Kansas
                          School of Medicine, Wichita, KS, USA
SOURCE:                   Arthritis Rheum. (2000), 43(2), 378-385
                          CODEN: ARHEAW; ISSN: 0004-3591
PUBLISHER:                Lippincott Williams & Wilkins
DOCUMENT TYPE:            Journal
LANGUAGE:                 English
AB   Objective: Because there is controversy regarding the efficacy of
     acetaminophen in rheumatic diseases and because apparently safer
     nonsteroidal antiinflammatory drugs (NSAIDs) are being produced, we
     surveyed rheumatic disease patients about their preferences for these
     agents to det. the degree to which one type of therapeutic agent is
     preferred over the other.  Methods: In 1998, we surveyed by mailed
     questionnaire 1,799 patients with osteoarthritis (OA), rheumatoid
```

arthritis, or fibromyalgia who were participating in a long-term outcome study. Patients who had taken acetaminophen rated the effectiveness of acetaminophen, compared its effectiveness with that of NSAIDs, and then rated their overall satisfaction with acetaminophen compared with NSAIDs when both effectiveness and side effects were considered. Results: Two-thirds of study participants had taken acetaminophen. About 37% of patients who had taken acetaminophen found it to be moderately or very effective and about 63% indicated that it was not effective or was only slightly effective. One-fourth of the patients found acetaminophen and NSAIDs to be equally effective, but >60% found acetaminophen to be much less effective or somewhat less effective. About 12% preferred acetaminophen to NSAIDs. When both effectiveness and side effects were considered together, 25% of the patients had no preference, 60% preferred NSAIDs, and 14% preferred acetaminophen. Conclusion: There was a considerable and statistically significant preference for NSAIDs compared with acetaminophen among the 3 groups of rheumatic disease patients. Although this preference decreased slightly with age and was less pronounced in OA patients, the preference was noted among all categories of patients and was not altered by disease severity. If safety and cost are not issues, there would hardly ever be a reason to recommend acetaminophen over NSAIDs, since patients generally preferred NSAIDs and fewer than 14% preferred acetaminophen. If safety and costs are issues, then the recommendation of the American College Rheumatol. that acetaminophen be tried first seems correct, since 38.2% found acetaminophen to be as effective or more effective than NSAIDs.

REFERENCE COUNT: 49

REFERENCE(S):
(5) Altman, R; J Rheumatol 1998, V25, P2203 CAPLUS
(20) Hawkey, C; Lancet 1999, V353, P307 CAPLUS
(29) Jones, A; Ann Rheum Dis 1996, V55, P829 CAPLUS
(31) Lipsky, L; J Rheumatol 1998, V25, P2298 CAPLUS
(32) Lohmander, L; Ann Rheum Dis 1996, V55, P424 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

ST acetaminophen nonsteroidal **antiinflammatory** drug osteoarthritis **fibromyalgia**; rheumatoid arthritis acetaminophen nonsteroidal antiinflammatory drug; nonsteroidal antiinflammatory drug acetaminophen rheumatic disease

IT Muscle, disease
(**fibromyalgia**; preference for nonsteroidal **antiinflammatory** drugs over acetaminophen by rheumatic disease patients)

L23 ANSWER 2 OF 40 MEDLINE

Full-text

ACCESSION NUMBER: 94198863 MEDLINE
DOCUMENT NUMBER: 94198863 PubMed ID: 8148849
TITLE: [Value of the self-evaluation of functional and painful disorders for the differentiation between **fibromyalgia** and **inflammatory** rheumatic diseases].
Interet de l'auto-evaluation des troubles fonctionnels et douloureux pour la distinction entre **fibromyalgie** et rhumatisme **inflammatoire**.
AUTHOR: Renoux M; Hilliquin P; Menkes C J
CORPORATE SOURCE: Service de Rhumatologie A, Hopital Cochin, Paris.
SOURCE: REVUE DU RHUMATISME. EDITION FRANCAISE, (1993 Jul-Sep) 60 (7-8) 499-503.
Journal code: BQU; 9315664.
PUB. COUNTRY: France
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: French
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199405
ENTRY DATE: Entered STN: 19940523
Last Updated on STN: 19940523
Entered Medline: 19940511

AB The authors sought to determine whether the self-report questionnaire developed by L.F. Callahan and T. Pincus is of use for the diagnosis of fibromyalgia when severe and/or chronic pain raises doubts as to an inflammatory rheumatic disease. This questionnaire evaluates the ratio between pain severity assessed on a visual analog scale and impairment of activities of daily living. High values suggest fibromyalgia, whereas low values occur in rheumatoid arthritis patients. The French translation of the questionnaire was completed by 15 **fibromyalgia** patients and 22 patients with **inflammatory** arthritic syndrome of whom 15 had rheumatoid arthritis. Mean pain/impairment ratio was 5.85 +/- 0.68 (SEM) in the

fibromyalgia group versus 3.01 +/- 0.38 in the inflammatory rheumatism group. This difference was highly significant ($p = 0.001$ by Student's t test). The rheumatoid arthritis subgroup was also significantly different from the fibromyalgia group ($p = 0.003$). These findings are very similar to those reported by Callahan and Pincus. Our data confirm the value of this simple self-evaluation tool. In practice, ratios greater than 5 suggest fibromyalgia whereas ratios under 3 support the diagnosis of rheumatoid arthritis.

L23 ANSWER 3 OF 40 CAPLUS COPYRIGHT 2001 ACS
Full-text
ACCESSION NUMBER: 1999:593371 CAPLUS
DOCUMENT NUMBER: 132:91472
TITLE: Biological markers of fibromyalgia
AUTHOR(S): Maes, Michael
CORPORATE SOURCE: Clinical Research Center for Mental Health, University Department of Psychiatry, Antwerp, 2060, Belg.
SOURCE: Keio Univ. Symp. Life Sci. Med. (1999), 3(Somatoform Disorders), 111-121
CODEN: KUSMF9
PUBLISHER: Springer-Verlag Tokyo
DOCUMENT TYPE: Journal; General Review
LANGUAGE: English
AB A review with 89 refs. Fibromyalgia is a chronic condition characterized by widespread musculoskeletal pain and pressure hyperalgesia at characteristic sites, i.e., soft tissue tender points. The biophysiol. of fibromyalgia, however, has remained elusive. This paper reviews recent biol. research on the role of the neuroendocrine and immune systems in the biophysiol. of fibromyalgia. It is suggested that fibromyalgia is detd. by a combination of different pathophysiol. mechanisms, which reside in the catecholaminergic and immune systems and in peptidase activities. Subsensitive platelet α2-adrenoceptors suggest a lowered affinity of presynaptic receptors and thus could indicate a lower autoinhibitory activity on the catecho-laminergic neuron. The results do not corroborate the hypotheses that fibromyalgia is accompanied by a deficiency in serotonergic metab. or in disturbances in the hypothalamic-pituitary-adrenal axis. It is hypothesized that aberrant pain perception and depressive symptoms in fibromyalgia may result from decreases in prolyl endopeptidase (PEP, EC 3.4.21.26), a cytosolic endopeptidase which inactivates algesic (e.g., bradykinin, substance P) and depression-related peptides. Most results show no significant signs of **inflammation** in **fibromyalgia**, but show indications of immunosuppression.
REFERENCE COUNT: 89
REFERENCE(S): (3) Bhoola, K; Pharmacol Rev 1992, V44, P1 CAPLUS
(4) Bluthe, R; Ann NY Acad Sci 1992, V650, P268 CAPLUS
(9) Cunha, F; Br J Pharmacol 1992, V107, P660 CAPLUS
(10) Czlonkowski, A; Eur J Pharmacol 1993, V242, P229 CAPLUS
(11) DeLeo, J; J Interf Cytokine Res 1996, V16, P695 CAPLUS
ALL CITATIONS AVAILABLE IN THE RE FORMAT

L23 ANSWER 4 OF 40 MEDLINE
Full-text
ACCESSION NUMBER: 2001463229 IN-PROCESS
DOCUMENT NUMBER: 21399331 PubMed ID: 11508596
TITLE: Previous pain experience in women with **fibromyalgia** and **inflammatory** arthritis and nonpainful controls.
AUTHOR: Poyhia R; Da Costa D; Fitzcharles M A
CORPORATE SOURCE: Department of Medicine and Clinical Epidemiology, McGill University Health Center, McGill University, Montreal, Quebec, Canada.
SOURCE: JOURNAL OF RHEUMATOLOGY, (2001 Aug) 28 (8) 1888-91.
Journal code: JWX; 7501984. ISSN: 0315-162X.
PUB. COUNTRY: Canada
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: IN-PROCESS; NONINDEXED; Priority Journals
ENTRY DATE: Entered STN: 20010820
Last Updated on STN: 20010820
AB OBJECTIVE: To examine the frequency of commonly occurring pain and adverse experiences throughout life by self-report in women with **fibromyalgia** (FM) and chronic **inflammatory** arthritis (IA) and nonpainful healthy women. METHODS: Fifty-one patients with FM and 44 with IA and 52 nonpainful healthy controls were consecutively interviewed in a tertiary

clinic setting regarding the occurrence of lifetime common pain experience and adverse events, as well as a family history of FM and/or a childhood pain environment. RESULTS: Patients with FM reported significantly more irritable bowel syndrome, migraine headaches, severe menstrual pain, physical and psychological trauma affecting well being, family history of FM, and family pain environment than subjects with IA or controls. Both patient groups had more adult hospitalizations and surgeries than the controls. CONCLUSION: Patients with FM report a high rate of varied pain and adverse experiences throughout life. This real or perceived experience of pain supports the concept that FM is a lifetime disorder of pain processing.

L23 ANSWER 5 OF 40 MEDLINE
Full-text
ACCESSION NUMBER: 1999272911 MEDLINE
DOCUMENT NUMBER: 99272911 PubMed ID: 10341365
TITLE: The immune-**inflammatory** pathophysiology of **fibromyalgia**: increased serum soluble gp130, the common signal transducer protein of various neurotrophic cytokines.
AUTHOR: Maes M; Libbrecht I; Van Hunsel F; Lin A H; De Clerck L; Stevens W; Kenis G; de Jongh R; Bosmans E; Neels H
CORPORATE SOURCE: University Department of Psychiatry, Clinical Research Center for Mental Health (CRC-MH), Antwerp, Belgium.. m.maes@unicall.be
SOURCE: PSYCHONEUROENDOCRINOLOGY, (1999 May) 24 (4) 371-83. Journal code: QGC; 7612148. ISSN: 0306-4530.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199907
ENTRY DATE: Entered STN: 19990727
Last Updated on STN: 19990727
Entered Medline: 19990715
AB Fibromyalgia is a chronic, painful musculoskeletal disorder characterized by widespread pain, pressure hyperalgesia, morning stiffness and by an increased incidence of depressive symptoms. The etiology, however, has remained elusive. The aim of the present study was to examine the **inflammatory** response system (IRS) in **fibromyalgia**. Serum interleukin-6 (IL-6), soluble IL-6 receptor (sIL-6R), sgp130, sIL-1R antagonist (IL-1RA) and sCD8 were determined in 33 healthy volunteers and in 21 fibromyalgia patients, classified according to the American College of Rheumatology criteria. Severity of illness was measured with several pain scales, dolorimetry and the Hamilton Depression Rating Scale (HDRS). Serum sgp130 was significantly higher and serum sCD8 significantly lower in fibromyalgia patients than in healthy volunteers. Serum sIL-6R and sIL-1RA were significantly higher in fibromyalgia patients with an increased HDRS score (> or = 16) than in normal volunteers and fibromyalgia patients with a HDRS score < 16. In fibromyalgia patients, an important part of the variance in sCD8 (50.3%) and IL-1RA (19.3%) could be explained by the HDRS score; 74.3% of the variance in sIL-6R was explained by the combined effects of pain symptoms and the HDRS score; and 25.9% of the variance in serum sgp130 was explained by stiffness. The results support the contention that pain and stiffness in fibromyalgia may be accompanied by a suppression of some aspects of the IRS and that the presence of clinically significant depressive symptoms in fibromyalgia is associated with some signs of IRS activation.

=> s l23 not py>1997
L24 19 L23 NOT PY>1997

=> d ibib abs kwic 1-5

L24 ANSWER 1 OF 19 MEDLINE
Full-text
ACCESSION NUMBER: 97455900 MEDLINE
DOCUMENT NUMBER: 97455900 PubMed ID: 9310112
TITLE: Dermal IgG deposits and increase of mast cells in patients with fibromyalgia--relevant findings or epiphenomena?.
AUTHOR: Enestrom S; Bengtsson A; Frodin T
CORPORATE SOURCE: Department of Pathology, Linkoping University, Sweden.
SOURCE: SCANDINAVIAN JOURNAL OF RHEUMATOLOGY, (1997) 26 (4) 308-13. Journal code: UD1; 0321213. ISSN: 0300-9742.
PUB. COUNTRY: Norway

Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199710
ENTRY DATE: Entered STN: 19971105
Last Updated on STN: 19971105
Entered Medline: 19971023

AB Skin biopsies from 25 patients with fibromyalgia, 5 healthy controls, 8 patients with rheumatoid arthritis, and 9 patients with local chronic pain after whiplash injury, were examined for the occurrence of IgG deposits and collagen types, using direct and indirect immunofluorescence, and for dermal connective tissue mast cells, using semithin Epon sections. Fibromyalgia skin biopsies had significantly higher values of IgG deposits in the dermis and vessel walls and showed a higher reactivity for collagen III. They also had a higher mean number of mast cells. There was a correlation between the percentage of damaged/degranulated mast cells and the individual IgG immunofluorescence scores. These findings support the hypothesis of neurogenic **inflammation** involvement in **fibromyalgia**.

L24 ANSWER 2 OF 19 MEDLINE
Full-text
ACCESSION NUMBER: 97373032 MEDLINE
DOCUMENT NUMBER: 97373032 PubMed ID: 9229086
TITLE: An outcome analysis of 100 women after explantation of silicone gel breast implants.
COMMENT: Comment in: Ann Plast Surg. 1998 Jan;40(1):103-5
Comment in: Ann Plast Surg. 1998 Jul;41(1):101-3
AUTHOR: Peters W; Smith D; Fornasier V; Lugowski S; Ibanez D
CORPORATE SOURCE: Division of Plastic Surgery, University of Toronto, Ontario, Canada.
SOURCE: ANNALS OF PLASTIC SURGERY, (1997 Jul) 39 (1) 9-19.
Journal code: 5VB; 7805336. ISSN: 0148-7043.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199708
ENTRY DATE: Entered STN: 19970902
Last Updated on STN: 19990129
Entered Medline: 19970819

AB A prospective outcome analysis was conducted on 100 consecutive women who requested explantation of their silicone gel breast implants from January 6, 1992 (the moratorium), through 1995. Eighteen patients were referred by rheumatologists with a diagnosis of autoimmune or rheumatic disease. Six had autoimmune disease (systemic lupus, 2 patients; rheumatoid arthritis, 2 patients; multiple sclerosis, 1 patient; and Raynaud's disease, 1 patient). Twelve had rheumatic disease (**fibromyalgia**, 10 patients; **inflammatory** arthritis, 2 patients). All of these 18 patients had developed symptoms of their disease after they had received implants. All 100 patients were extensively evaluated pre- and postoperatively by interviews, clinical assessment, and by assay of the following laboratory tests: rheumatoid factor, ESR, ANA, and anti-Ro/SSA, -La/SSP, -Sm, -RNP, -double-stranded deoxyribonucleic acid, -Scl-70, -centromere, and -cardiolipin. Patients were also evaluated by a questionnaire that was sent at a mean time of 2.7 years postexplantation (range, 1-5 years), which had a 75% response rate. Reasons for implants were augmentation, 75%; lifting, 11%; reconstruction, 12%; and congenital aplasia, 2%. The mean age at first implant was 28.9 years (range, 13-55 years) and at explantation was 41.5 years (range, 25-65 years). The mean duration of implantation was 12.0 years (range, 1-27 years). Thirty-six percent of the patients had undergone at least one closed capsulotomy and 54% at least one open capsulotomy. The mean reasons for explantation were suspected silicone-related health problems, 76%; suspected rupture, 59%; breast firmness, 36%; breast pain, 36%; and musculoskeletal pain, 23%. Before explantation 75% of the questionnaire respondees had lost some sensitivity in their nipples following their breast augmentation. In 36% of those 75 patients, that loss was almost complete. Loss of sensitivity was related to capsular contracture and to pain ($p < 0.05$). Following explantation there was significant improvement in nipple sensitivity in 38% of breasts in the 75 respondees. A total of 186 implants were removed. Fifty-seven percent had failed by rupturing or leaking. Only 3.2% demonstrated extravasation extracapsularly. Twenty-five percent of the capsules were calcified, demonstrating visible plaques of calcification on their inner surface. Forty-two percent were colonized by bacteria. The prevalence of class III-IV capsular contracture was 61% and it was related to implant

location, duration in situ, and capsular calcification ($p < 0.05$), but not to capsular colonization or implant integrity ($p > 0.05$). Only 43 of the 100 patients elected to have saline implants inserted. Of the others, 56% felt that the shell of the saline implant could be associated with medical problems. The others felt that breast size was of minor importance to them at this time. There were few complications from the explantation procedure. Two "masses" were discovered-one was an occult carcinoma, the other a galactocele. There was one wound infection, which responded to antibiotics. Three patients developed decreased sensitivity and 3 developed increased breast pain. From the patient questionnaires, in those women who did not have saline implants inserted, 15% felt that their breast appearance was improved after explantation, 36% were "pleased," 33% were disappointed, and 13% felt "mutilated". In women who did have saline implants inserted, 18% felt that their breast appearance was now improved, 60% were "pleased," and 14% were disappointed, mainly because of wrinkling. At a mean time of 2.7 years (range, 1-5 years) after explantation, 45% of the 75 questionnaire respondees felt that their implants had caused permanent health problems and 56% felt that they had not been given adequate informed consent by their original surgeon (particularly regarding implant rupture and a possible relationship to medical disease). (ABSTRACT TRUNCATED)

L24 ANSWER 3 OF 19 MEDLINE
Full-text
ACCESSION NUMBER: 97068336 MEDLINE
DOCUMENT NUMBER: 97068336 PubMed ID: 8911646
TITLE: The three-way interactions between the hypothalamic-pituitary-adrenal and gonadal axes and the immune system.
AUTHOR: Torpy D J; Chrousos G P
CORPORATE SOURCE: Developmental Endocrinology Branch, National Institute of Child Health and Human Development, Bethesda, MD 20892, USA.
SOURCE: BAILLIERES CLINICAL RHEUMATOLOGY, (1996 May) 10 (2) 181-98. Ref: 102
Journal code: CRY; 8805770. ISSN: 0950-3579.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199702
ENTRY DATE: Entered STN: 19970305
Last Updated on STN: 19970305
Entered Medline: 19970218

AB The stress system is controlled by brain nuclei at the hypothalamus and brainstem. These nuclei interact with each other and control the HPA axis and sympathetic nervous systems, respectively. Major inputs to the stress system arise from the cerebral cortex and subcortical systems, the sensory organs and nerves, and the endocrine and immune systems. The major peripheral effectors of the stress system are glucocorticoids and the catecholamines. Pathological hypoactivity of the stress system has been associated with atypical depression, the chronic fatigue/**fibromyalgia** syndromes and autoimmune **inflammatory** disease; hyperactivity with melancholic depression and anxiety disorders. The stress system responds in a quantitatively and qualitatively specific fashion to different stressors. A major role of the HPA axis is to restrain the immune system and prevent tissue damage. Reciprocal interactions between the HPA axis and immune system constitutes a new endocrine feedback loop that has given rise to the field of neuroendocrine immunology. Gonadal axis hormones directly, and indirectly via the HPA axis, alter the tone of the immune system and the quality and quantity of the inflammatory responses. Effects of the HPA axis on the gonadal axis are consistent with conservation and redirection of valuable resources towards homeostasis during times of stress. These complex interactions between the HPA axis, immune and the gonadal systems may prove to be fundamental in the genesis and perpetuation of autoimmune disease.

L24 ANSWER 4 OF 19 MEDLINE
Full-text
ACCESSION NUMBER: 97027497 MEDLINE
DOCUMENT NUMBER: 97027497 PubMed ID: 8873636
TITLE: Neuroimmune mechanisms in health and disease: 2. Disease.
AUTHOR: Anisman H; Baines M G; Berczi I; Bernstein C N; Blennerhassett M G; Gorczynski R M; Greenberg A H; Kisil F

T; Mathison R D; Nagy E; Nance D M; Perdue M H; Pomerantz D K; Sabbadini E R; Stanisz A; Warrington R J
CORPORATE SOURCE: Department of Psychology, Carloton University, Ottawa, Ont.
SOURCE: CMAJ, (1996 Oct 15) 155 (8) 1075-82. Ref: 63
Journal code: CVV; 9711805. ISSN: 0820-3946.
PUB. COUNTRY: Canada
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English
FILE SEGMENT: Abridged Index Medicus Journals; Priority Journals
ENTRY MONTH: 199611
ENTRY DATE: Entered STN: 19961219
Last Updated on STN: 19980206
Entered Medline: 19961112

AB In the second part of their article on the emerging field of neuroimmunology, the authors present an overview of the role of neuroimmune mechanisms in defence against infectious diseases and in immune disorders. During acute febrile illness, immune-derived cytokines initiate an acute phase response, which is characterized by fever, inactivity, fatigue, anorexia and catabolism. Profound neuroendocrine and metabolic changes take place: acute phase proteins are produced in the liver, bone marrow function and the metabolic activity of leukocytes are greatly increased, and specific immune reactivity is suppressed. Defects in regulatory processes, which are fundamental to immune disorders and inflammatory diseases, may lie in the immune system, the neuro endocrine system or both. Defects in the hypothalamus-pituitary-adrenal axis have been observed in autoimmune and rheumatic diseases, chronic **inflammatory** disease, chronic fatigue syndrome and **fibromyalgia**. Prolactin levels are often elevated in patients with systemic lupus erythematosus and other autoimmune diseases, whereas the bioactivity of prolactin is decreased in patients with rheumatoid arthritis. Levels of sex hormones and thyroid hormone are decreased during severe inflammatory disease. Defective neural regulation of inflammation likely plays a pathogenic role in allergy and asthma, in the symmetrical form of rheumatoid arthritis and in gastrointestinal inflammatory disease. A better understanding of neuroimmunoregulation holds the promise of new approaches to the treatment of immune and inflammatory diseases with the use of hormones, neurotransmitters, neuropeptides and drugs that modulate these newly recognized immune regulators.

L24 ANSWER 5 OF 19 MEDLINE
Full-text
ACCESSION NUMBER: 94244236 MEDLINE
DOCUMENT NUMBER: 94244236 PubMed ID: 8187451
TITLE: Fibromyalgia associated with female urethral syndrome.
AUTHOR: Paira S O
CORPORATE SOURCE: Department of Internal Medicine, Hospital Jose M. Cullen, Santa Fe, Argentina.
SOURCE: CLINICAL RHEUMATOLOGY, (1994 Mar) 13 (1) 88-9.
Journal code: DI6; 8211469. ISSN: 0770-3198.
PUB. COUNTRY: Belgium
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199406
ENTRY DATE: Entered STN: 19940629
Last Updated on STN: 19940629
Entered Medline: 19940623

AB Thirty-eight out of 212 patients (18%) with fibromyalgia met the criteria for the definition of female urethral syndrome (FUS). None of the patients from the control group met these criteria. The treatment for FUS was the same as that for **fibromyalgia**: cyclobenzaprine or diazepam and nonsteroidal anti-**inflammatory** drugs with a partial response in both pathologies. We should consider FUS in the evaluation of every patient with fibromyalgia.

=> d ibib abs kwic 6-10

L24 ANSWER 6 OF 19 MEDLINE
Full-text
ACCESSION NUMBER: 94198863 MEDLINE
DOCUMENT NUMBER: 94198863 PubMed ID: 8148849
TITLE: [Value of the self-evaluation of functional and painful disorders for the differentiation between **fibromyalgia**

and **inflammatory** rheumatic diseases].
Interet de l'auto-evaluation des troubles fonctionnels et douloureux pour la distinction entre **fibromyalgie** et rhumatisme **inflammatoire**.
AUTHOR: Renoux M; Hilliquin P; Menkes C J
CORPORATE SOURCE: Service de Rhumatologie A, Hopital Cochin, Paris.
SOURCE: REVUE DU RHUMATISME. EDITION FRANCAISE, (1993 Jul-Sep) 60 (7-8) 499-503.
Journal code: BQU; 9315664.
PUB. COUNTRY: France
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: French
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199405
ENTRY DATE: Entered STN: 19940523
Last Updated on STN: 19940523
Entered Medline: 19940511

AB The authors sought to determine whether the self-report questionnaire developed by L.F. Callahan and T. Pincus is of use for the diagnosis of fibromyalgia when severe and/or chronic pain raises doubts as to an inflammatory rheumatic disease. This questionnaire evaluates the ratio between pain severity assessed on a visual analog scale and impairment of activities of daily living. High values suggest fibromyalgia, whereas low values occur in rheumatoid arthritis patients. The French translation of the questionnaire was completed by 15 **fibromyalgia** patients and 22 patients with **inflammatory** arthritic syndrome of whom 15 had rheumatoid arthritis. Mean pain/impairment ratio was 5.85 +/- 0.68 (SEM) in the fibromyalgia group versus 3.01 +/- 0.38 in the inflammatory rheumatism group. This difference was highly significant ($p = 0.001$ by Student's t test). The rheumatoid arthritis subgroup was also significantly different from the fibromyalgia group ($p = 0.003$). These findings are very similar to those reported by Callahan and Pincus. Our data confirm the value of this simple self-evaluation tool. In practice, ratios greater than 5 suggest fibromyalgia whereas ratios under 3 support the diagnosis of rheumatoid arthritis.

L24 ANSWER 7 OF 19 MEDLINE
Full-text
ACCESSION NUMBER: 93312134 MEDLINE
DOCUMENT NUMBER: 93312134 PubMed ID: 8323398
TITLE: Thyroid dysfunction in rheumatoid arthritis: a controlled prospective survey.
AUTHOR: Shiroky J B; Cohen M; Ballachey M L; Neville C
CORPORATE SOURCE: Division of Rheumatology, Montreal General Hospital, Quebec, Canada.
SOURCE: ANNALS OF THE RHEUMATIC DISEASES, (1993 Jun) 52 (6) 454-6.
Journal code: 62W; 0372355. ISSN: 0003-4967.
PUB. COUNTRY: ENGLAND: United Kingdom
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199308
ENTRY DATE: Entered STN: 19930813
Last Updated on STN: 19930813
Entered Medline: 19930803

AB OBJECTIVES--To determine whether thyroid dysfunction is found with increased frequency in patients with rheumatoid arthritis (RA). METHODS--A controlled prospective survey was conducted on a cohort of patients with RA derived from a hospital clinic and a private surburban rheumatology practice. A control group with similar demographic features was generated from the same sources and included subjects with either osteoarthritis or fibromyalgia. Consecutive patients were evaluated over a six month period. The evaluation included a complete history and physical examination, and determination of serum thyroxine, free thyroxine, triiodothyronine, thyroid stimulating hormone (IRMA), antinuclear antibodies, and rheumatoid factor. RESULTS--Of the 91 women with RA evaluated, 29 (30%) had evidence of thyroid dysfunction compared with 10 (11%) of 93 controls. The excess thyroid dysfunction is due to either hypothyroidism or Hashimoto's thyroiditis and was independent of age, increasing duration of disease, rheumatoid factor, and antinuclear antibodies. CONCLUSIONS--Thyroid dysfunction is seen at least three times more often in women with RA than in women with similar demographic features with non-**inflammatory** rheumatic diseases such as osteoarthritis and **fibromyalgia**.

L24 ANSWER 8 OF 19 MEDLINE

Full-text
ACCESSION NUMBER: 92007388 MEDLINE
DOCUMENT NUMBER: 92007388 PubMed ID: 1717231
TITLE: Alpha-like EEG activity in non-REM sleep and the fibromyalgia (fibrositis) syndrome.
AUTHOR: Horne J A; Shackell B S
CORPORATE SOURCE: Department of Human Sciences, Loughborough University, Leicestershire, U.K.
SOURCE: ELECTROENCEPHALOGRAPHY AND CLINICAL NEUROPHYSIOLOGY, (1991 Oct) 79 (4) 271-6.
Journal code: EEH; 0375035. ISSN: 0013-4694.
PUB. COUNTRY: Ireland
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199111
ENTRY DATE: Entered STN: 19920124
Last Updated on STN: 19960129
Entered Medline: 19911112

AB The occurrence and characteristics of alpha-like activity during non-REM (NREM) sleep were examined in 11 subjects suffering from non-**inflammatory** (non-rheumatoid) musculoskeletal pain--**fibromyalgia** ('fibrositis'), and in 15 symptom-free controls. Both groups claimed to be good sleepers. Mean percentage alpha-like activity in sleep stages 2, 3, 4 and for NREM as a whole were greatest for the fibromyalgia group, but not significantly different from those of the controls. Overlap in the distribution of NREM alpha-like activity in sleep between the two groups indicated that it is not directly related to musculoskeletal symptoms. Spectral analyses showed a frontal area prevalence of this (kappa?) activity in the fibromyalgia group.

L24 ANSWER 9 OF 19 MEDLINE
Full-text
ACCESSION NUMBER: 91194011 MEDLINE
DOCUMENT NUMBER: 91194011 PubMed ID: 2084244
TITLE: Absence of autoantibodies in primary fibromyalgia.
AUTHOR: Bengtsson A; Ernerudh J; Vrethem M; Skogh T
CORPORATE SOURCE: Linkoping University Hospital, Sweden.
SOURCE: JOURNAL OF RHEUMATOLOGY, (1990 Dec) 17 (12) 1682-3.
Journal code: JWX; 7501984. ISSN: 0315-162X.
PUB. COUNTRY: Canada
Journal; Article; (JOURNAL ARTICLE)
LANGUAGE: English
FILE SEGMENT: Priority Journals
ENTRY MONTH: 199105
ENTRY DATE: Entered STN: 19910602
Last Updated on STN: 19910602
Entered Medline: 19910516

AB Sera from patients with primary fibromyalgia (223 sera, 210 women; 13 men) were analyzed, by immunofluorescence microscopy, for the presence of antibodies directed against cell nuclei (ANA), smooth muscle, mitochondria and other tissue antigens present in cryostat sections of rat organs (liver, kidney and stomach). Sera from blood donors (255 sera, 75 women; 180 men) served as a comparative group. The occurrence of serum autoantibodies in patients with fibromyalgia did not differ significantly from the reference group. Our results differ from those of others, who have suggested a relation between **fibromyalgia** and **inflammatory** rheumatic diseases.

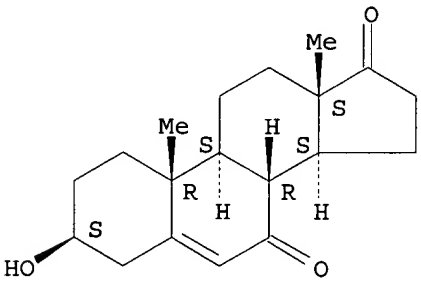
L24 ANSWER 10 OF 19 MEDLINE
Full-text
ACCESSION NUMBER: 90265919 MEDLINE
DOCUMENT NUMBER: 90265919 PubMed ID: 2189154
TITLE: Polymyalgia rheumatica.
AUTHOR: Cohen M D; Ginsburg W W
CORPORATE SOURCE: Division of Rheumatology and Internal Medicine, Mayo Clinic Jacksonville, Florida.
SOURCE: RHEUMATIC DISEASES CLINICS OF NORTH AMERICA, (1990 May) 16 (2) 325-39. Ref: 64
Journal code: RDC; 8708093. ISSN: 0889-857X.
PUB. COUNTRY: United States
Journal; Article; (JOURNAL ARTICLE)
General Review; (REVIEW)
(REVIEW, TUTORIAL)
LANGUAGE: English

FILE SEGMENT: Priority Journals
ENTRY MONTH: 199006
ENTRY DATE: Entered STN: 19900810
Last Updated on STN: 19900810
Entered Medline: 19900629

AB Polymyalgia rheumatica is a syndrome that occurs in the elderly and is characterized by pain and stiffness involving the neck, the shoulder girdle, and the hip girdle. The aching should be present for greater than one month. Polymyalgia rheumatica may be more common than reported. The etiology remains unknown. There is generally little found pathologically in this disease. The physical examination is often not impressive. Synovitis may be a main contributing factor to many of the symptoms seen in patients with polymyalgia rheumatica. Symptoms often do not correlate with physical findings. Polymyalgia rheumatica must be differentiated from many conditions since the diagnosis remains entirely clinical. Osteoarthritis, flu syndromes, **inflammatory** myopathies, **fibromyalgia**, and depression all have features that may mimic polymyalgia rheumatica. Malignancies and infections may also be difficult to separate from polymyalgia rheumatica. Polymyalgia rheumatica may also be extremely difficult to differentiate from seronegative rheumatoid arthritis in patients older than 50 years. Although some patients with polymyalgia rheumatica have underlying giant cell arteritis, the majority apparently do not. The distinction between polymyalgia rheumatica and giant cell arteritis cannot be made on the basis of laboratory studies and relies solely on clinical symptoms and physical findings. Although nonsteroidal antiinflammatory medications may control symptoms in patients with mild disease, most patients with polymyalgia rheumatica require low-dose corticosteroids. The tapering schedule for the corticosteroids is contingent upon the response of symptoms and laboratory parameters. Polymyalgia rheumatica usually follows a benign course with almost complete response to an adequate treatment program. Recently, there have been several studies suggesting that the course of polymyalgia rheumatica may not be as short and simple as once proposed. Nevertheless, many patients may be completely weaned from corticosteroids. Other agents have been used in this disease, but for the most part their use remains somewhat controversial. Patients must be monitored carefully. Most patients do well, and treatment is effective.

L3 ANSWER 5 OF 5 REGISTRY COPYRIGHT 2001 ACS
RN 566-19-8 REGISTRY
CN Androst-5-ene-7,17-dione, 3-hydroxy-, (3.beta.)- (9CI) (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN Androst-5-ene-7,17-dione, 3.beta.-hydroxy- (8CI)
OTHER NAMES:
CN 3.beta.-Hydroxy-5-androstene-7,17-dione
CN 5-Androsten-3.beta.-ol-7,17-dione
CN 7-Ketodehydroepiandrosterone
CN 7-Oxodehydroepiandrosterone
FS STEREOSEARCH
MF C19 H26 O3
LC STN Files: BEILSTEIN*, BIOSIS, CA, CAOLD, CAPLUS, CASREACT, IFICDB, IFIPAT, IFIUDB, MEDLINE, TOXLIT, USPATFULL
(*File contains numerically searchable property data)

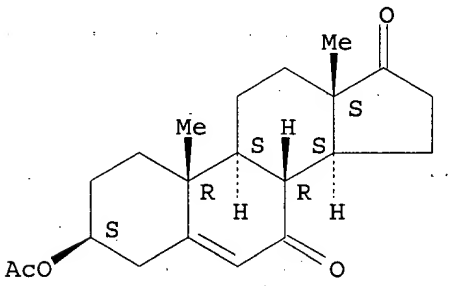
Absolute stereochemistry.



63 REFERENCES IN FILE CA (1967 TO DATE)
5 REFERENCES TO NON-SPECIFIC DERIVATIVES IN FILE CA
63 REFERENCES IN FILE CAPLUS (1967 TO DATE)
2 REFERENCES IN FILE CAOLD (PRIOR TO 1967)

L4 ANSWER 3 OF 5 REGISTRY COPYRIGHT 2001 ACS
RN 1449-61-2 REGISTRY
CN **Androst-5-ene-7,17-dione, 3-(acetyloxy)-, (3.beta.)- (9CI)** (CA INDEX NAME)
OTHER CA INDEX NAMES:
CN **Androst-5-ene-7,17-dione, 3.beta.-hydroxy-, acetate (6CI, 7CI, 8CI)**
OTHER NAMES:
CN **3.beta.-Acetoxy-5-androsten-7,17-dione**
CN **3.beta.-Acetoxyandrost-5-ene-7,17-dione**
CN **5-Androsten-3.beta.-ol-7,17-dione acetate**
FS STEREOSEARCH
MF C21 H28 O4
LC STN Files: BEILSTEIN*, CA, CAOLD, CAPLUS, CASREACT, CHEMCATS, CSCHEM, IFICDB, IFIPAT, IFIUDB, TOXLIT, USPATFULL
(*File contains numerically searchable property data)

Absolute stereochemistry.



49 REFERENCES IN FILE CA (1967 TO DATE)
49 REFERENCES IN FILE CAPLUS (1967 TO DATE)
14 REFERENCES IN FILE CAOLD (PRIOR TO 1967)